Cortical Reorganization Following Psychoeducational Counselling and Residual Inhibition Therapy (RIT) in Individuals with Tinnitus

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Int Arch Otorhinolaryngol 2022;26(4):e701–e711.

Abstract

Introduction Psychoeducational counselling and residual inhibition therapy (RIT) are traditional approaches used in many clinics to manage tinnitus. However, neurophysiological studies to evaluate posttreatment perceptual and functional cortical changes in humans are scarce.

Objectives The present study aims to explore whether cortical auditory-evoked potentials (CAEPs; N1 and P3) reflect the effect of modified RIT and psychoeducational counselling, and whether there is a correlation between the behavioral and electrophysiological measures.

Methods Ten participants with continuous and bothersome tinnitus underwent a session of psychoeducational counselling and modified RIT. Perceptual measures and CAEPs were recorded pre- and posttreatment. Further, the posttreatment measures were compared with age and gender-matched historical control groups.

Results Subjectively, 80% of the participants reported a reduction in the loudness of their tinnitus. Objectively, there was a significant reduction in the posttreatment amplitude of N1 and P3, with no alterations in latency. There was no correlation between the perceived difference in tinnitus loudness and the difference in P3 amplitude (at Pz).

Conclusion The perceptual and functional (as evidenced by sensory, N1, and cognitive, P3 reduction) changes after a single session of RIT and psychoeducational counselling are suggestive of plastic changes at the cortical level. The current study serves as preliminary evidence that event-related potentials (ERPs) can be used to quantify the physiological changes that occur after the intervention for tinnitus.

Keywords
- residual inhibition therapy
- tinnitus
- psychoeducational counselling
- treatment
- cortical reorganization

Introduction

Tinnitus is a heterogeneous condition prevalent across the globe. It affects approximately one in ten adults.[1] The diverse causes of tinnitus make it difficult to narrow down to a single mechanism. A variety of mechanisms targeting the auditory periphery to the central level has been postulated. A definitive cure for tinnitus is still a pipe dream for researchers worldwide. Traditional management techniques aim at either eliminating tinnitus or reducing the individual's

received
December 24, 2020
accepted after revision
August 22, 2021
published online
June 14, 2022

DOI https://doi.org/10.1055/s-0042-1743287.
ISSN 1809-9777.

Thieme Revinter Publicações Ltda., Rua do Matoso 170, Rio de Janeiro, RJ, CEP 20270-135, Brazil

response to it.[2] These methods include tinnitus retraining therapy (TRT), cognitive behavioral therapy (CBT), sound therapy, neuromodulation, relaxation therapy, educational counselling, residual inhibition therapy (RIT), yoga etc.

Counselling, a cornerstone in tinnitus treatment, will empower individuals to de-attend to their tinnitus and eventually get habituated to it.[3] There is a strong relationship between tinnitus and psychological symptoms like anxiety, distress, and depression, which highlights the importance of a psychoeducational approach in counselling. The more knowledge an individual holds about his or her condition, the better the therapeutic outcomes.[4] Psychoeducational counselling aids in correcting maladaptive thoughts and behaviors by exploring the problem and clarifying the purpose and expected outcome of the intervention.[5] Education alone may be sufficient for some individuals. However, for some individuals an education on tinnitus solely can be sufficient.[6,7]

A renowned and commonly used therapy in many clinics, RIT involves the use of external noise to alter the patients' perception or reaction to tinnitus. Residual inhibition (RI, proposed by James Spalding in 1903) is a phenomenon in which there is a temporary quieting of the tinnitus after listening to a trigger sound. Acoustic stimulation can be provided using a variety of stimuli ranging from white noise, pure tones or noise that match an individuals tinnitus frequency, notched noise and filtered music. The effect of RI can last from a second to several hours, depending on the duration of the masking sound.[8,9] Temporary suppression/desensitization of neuronal excitation[10] and the action of metabotropic glutamate receptors (mGluRs) in inferior colliculus (IC) neurons[11] have been postulated as underlying reasons for the effectiveness of RI. However, the neural mechanism underlying RI and its neurophysiological correlates in humans are yet to be completely understood. Approximately 80% of individuals suffering from tinnitus are known to have some degree of RI,[12–14] which tends to last one minute on average. [15] However, there are also studies[10] that report contradictory findings, with either little or no improvement in tinnitus on any of their participants. The provision of RIT using frequency-specific narrow-band noise, at 10dBSL for 60 seconds, was questionable, and a modification in its paradigm has been suggested.[10] It has been shown that the duration, the intensity, and the sound spectrum used to induce RI influence its magnitude and duration.[16] Hence, in the present study, a modified RI paradigm was incorporated to intensify the treatment results.

The effectiveness of any tinnitus therapy can be determined by either subjective or objective measures. The most commonly-used subjective measures are questionnaires (such as the Tinnitus Handicap Inventory [THI], the Tinnitus Functional Index [TFI], and the Tinnitus Questionnaire [TQ]) and scales (such as the Likert scale and the Visual Analogue Scale [VAS]). They are mostly patient- or client-centered.

With cortical auditory-evoked potentials (CAEPs), on the other hand, physicians examine functional changes in the cortical processing using an external stimulus. The early waves or components peaking roughly within the first 200 ms after the onset of the stimulus reflect the sensory processing, and are termed exogenous components (P1, N1, P2 and N2). Event-related potentials (ERPs) generated in later parts reflect the "cognitive" process, and are termed as "endogenous" ERPs (P3, N4 etc.). P3 is a cognitive potential that peaks around 250 ms and 400 ms poststimulus for most adults between 20 and 70 years of age. It has a topographic maximum distribution at frontoparietal regions linking to attention and memory processes.[17–19] There are diverse regions that contribute to the generation of P3, including the hippocampus, temporal, frontal, and parietal cortical areas, structures within the limbic system, and the thalamus. P3 is reported to be altered in individuals with tinnitus, which indicates attentional dysfunction.[20–25]

The literature on CAEPs in individuals with tinnitus reports varied results. There have been shreds of evidence that tinnitus affects either or both the sensory and cognitive components of CAEPs, suggesting a difference in the way sound is being processed.[20–28] In vitro animal studies[16] have shown that the hyperactive neural firings decrease as a result of RIT. However, there are no neurophysiological studies reported on humans following RIT or educational counselling.

Aim of the Study

The present study aims at evaluating the perceptual and functional cortical changes following a session of modified RIT and psychoeducational counselling using auditory components N1 and P3.

Objectives

1. To evaluate the perceptual changes after a single session of tinnitus treatment.
2. To evaluate the neurophysiological changes associated with the perceptual changes using CAEPs.
3. To observe the correlation or lack thereof between the perceptual and functional cortical changes.

Materials and Method

The present study included 10 (5 female and 5 male) participants aged between 20 and 50 years with continuous and bothersome tinnitus lasting for 3 months or more. In total, 3 patients had bilateral tinnitus (subjects [SUB] 02, 07, and 09), 4 had tinnitus in their left ear (SUBs 01, 04, 08, and 10), and the remaining 3 (SUBs 03, 05 and 06), in the right ear. Detailed information regarding the participants is provided in ►**Table 1**. The study was approved by the institutional research committee and ethics committee (IEC516/2017). Written consent was obtained from all the participants before the initiation of the study. The present is the continuation of a previous study[25] which compared individuals with tinnitus to an age-, gender- and hearing-matched control group; the participants in the tinnitus group of the previous analysis formed the case group of the present study. To check if they could be included in the study, all participants

Table 1 Participants characteristics

Participant	Tinnitus laterality	Duration of tinnitus (in months)	Tinnitus pitch and loudness	THI Score	THI rating
SUB01	Left	48	4 kHz, 40 dBHL	42	Moderate
SUB02	Bilateral (right > left)	24	6 kHz; right – 50 dBHL; left – 10 dBHL	22	Mild
SUB03	Right	03	8 kHz, 40 dBHL	80	Catastrophic
SUB04	Left	12	8 kHz, 50 dBHL	14	Slight
SUB05	Right	12	1.5 kHz, 65 dBHL	24	Mild
SUB06	Right	06	4 kHz, 85 dBHL	32	Mild
SUB07	Bilateral (left > right)	Left: 120 Right: 02	4 kHz; right: 65 dBHL; left:70 dBHL	24	Mild
SUB08	Left	03	750 Hz, 100 dBHL	24	Mild
SUB09	Bilateral (right > left)	05	1 kHz; right: 35 dBHL; left: 30 dBHL	10	Slight
SUB10	Left	30	500 Hz, 70 dBHL	32	Mild

Abbreviations: SUB, subject; THI, Tinnitus Handicap Inventory.

underwent audiological and tinnitus evaluations performed using the Madsen-Astera (ANSI S3.43–1996) double-channel diagnostic audiometer coupled with TDH-50P (Telephonics Corporation DBA Communications System Division, New York, United States) headphones and Radio ear B-71 bone vibrator (Radio Ear, Denmark). Preliminary measures like tinnitus pitch and loudness matching were performed using a two-alternative forced-choice method. The participants' hearing thresholds were lower than 40 dBHL and they scored at least a minimal degree of handicap (that is, grade I), as indexed by the THI.[29] In addition to the aforementioned, they were asked to estimate the magnitude of their tinnitus loudness on a scale of 1 to 10, in which 10 corresponded to "extremely loud". The magnitude score before the actual testing served as the baseline for the subjective evaluation of tinnitus loudness.

Before the ERP Recording

Compumedics Neuroscan system (SCAN 4.5, Charlotte, USA) with a standard 32 channel EazyCapTM and combined mastoid reference was used to acquire the ERPs. Ocular potentials were acquired using the vertical electrooculogram (VEOG) and horizontal electrooculogram (HEOG) channels. Electrode impedance was ensured below 5 KΩ. The ERPs were recorded using a randomized auditory odd-ball paradigm with 1,000 Hz and 1,500 Hz pure tones serving as frequent and rare tones respectively. A total of 100 stimuli (at 75dB SPL over calibrated ER3A insert earphones) with frequent to rare ratios of 80:20 and interstimulus interval [ISI] of 1,000 ms were presented. The responses obtained were filtered using a band-pass filter with a high-pass and low -pass cut off at 1Hz and 30Hz respectively at a sampling rate of 500Hz. The participants were instructed to quietly count the number of rare stimuli and reveal them at the end of the testing. At the end of the recording session, the electrode connector was carefully detached, and the cap was retained on the participant's head for the rest of the procedure to prevent electrode migration or bridging.

Psychoeducational Counselling

Following the recording of ERPs (pre), all participants underwent a session of non-standardized structured psychoeducational counseling for tinnitus management, which lasted ~ 30 minutes and was provided by an experienced and qualified audiologist.[3] The counselling focused on the anatomy and physiology of the human ear, the audiological test results of the patients, the tinnitus and its causes, habituation, the role of attention in tinnitus, possible management options, RIT, and the procedure. The participants were also informed that there could be no benefit, or the benefits of RIT might be temporary. The main reason behind the counseling session was to make the participants aware of tinnitus and its causes, to reduce the fear and anxiety related to tinnitus by answering their questions, and to list the treatment options available.

Residual Inhibition Therapy (RIT)

The counselling session was followed by a session of modified RIT in an audiometric double room. In the modified RIT, all three parameters (intensity, duration, and spectrum) of the stimulus were modified to intensify the magnitude of tinnitus reduction and the duration of the RI. The participants were provided with white noise at a suprathreshold level (60 dBSL) for 2 minutes, which served as an acoustic enricher. We ensured that the stimulus did not reach an uncomfortable level for the participants. White noise was provided using the Madsen-Astera audiometer coupled with TDH-50P Headphones. Following the RIT, the patients were asked two questions: Does the tinnitus sound the same now? If not, how does it differ? The responses of all the participants were recorded; further, they were also asked to estimate a percentage for the change noted following the RIT. This estimated percentage served as the postsubjective measure.

Measures after the ERP

Following the RIT session, the participants underwent the ERP testing once again after 40 minutes to 1 hour. The test setting and paradigm remained constant. The impedance was tested for the second time, and was ensured to be lower than 5 KΩ.

Historical Control Group

A historical control group[25] containing ten individuals matched for age and gender was used to compare the functional cortical changes after a single session of modified RIT and psychoeducational counselling. The participants in the control group had no history of tinnitus or a diagnosis of neurological or psychiatric problems.

ERP Preprocessing and Analysis

The ERP data recorded were pre-processed using EEGLab (open source), version 14.1.1b,[30] which runs in Matlab R2017a (The MathWorks, Inc., Natick, MA, US). The data was preprocessed by providing channel locations using the BESA 4 Shell sphere model and manually interpolating or removing bad channels and blocks with any ocular or muscular artifact. The obtained waveforms were placed into two bins named frequent and infrequent. An epoch was run across each bin with a time window of 800ms and a prestimulus duration of −200ms. Independent component analysis (ICA) was performed regarding the epochs, followed by automated artifact rejections with the help of multiple artifact rejection algorithms (MARA)[31] to correct for ocular artifacts. Finally the epoched data was common referenced to the mastoid and the rand average waveform was computed using the ERPLab module in MATLAB (version 6.14). The peak latency and absolute amplitude of the sensory components (N1 and P2) were obtained from the frequent waveform, and that of the cognitive component (P3), from the rare waveform. The latency and amplitude of the sensory components were computed from two electrode sites (Fz and Cz), while, for the cognitive component, three midline electrode sites were considered (Fz, Cz, and Pz). The statistical analysis was performed using the Statistical Package for the Social Sciences (IBM SPSS Statistics for Windows, IBM Corp., Armonk, NY, US), version 20.1. The pre- and posttreatment data were subjected to normality testing using the Shapiro-Wilk test, and were found to be normally distributed. Repeated measures analysis of variance (ANOVA) (two levels of within-subject factor [electrodes Fz and Cz] for the N1 and P2 analysis, and three levels of within-subject factor [electrodes Fz, Cz, and Pz] were compared between the groups [pre- and posttreatment]) to compare the latency and amplitude of N1, P2, and P3. A simplified flowchart of the methodology is shown in ►**Fig. 1**.

Results

The mean age of the individuals with tinnitus was 38.8 (± 10.27) years. The mean pure-tone average (PTA) of all participants was 17.42 (±5.89) dBHL in the right ear, and 19.53 (±8.36) dBHL in the left ear. All participants had bothersome tinnitus with at least a grade-I handicap as indexed using the THI.

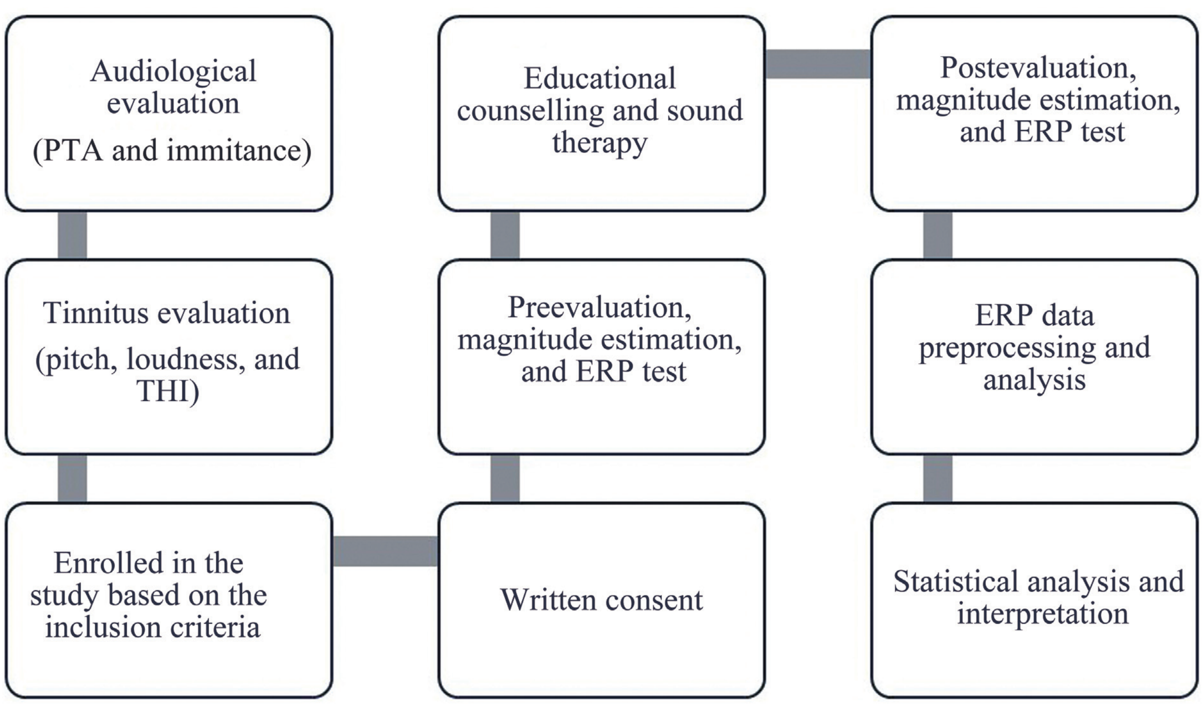

Fig. 1 Simplified flowchart of methodology of the present study.

Table 2 Subjective outcome measures of the individual participants

Participant	Tinnitus laterality	THI rating	Perceived reduction in tinnitus
SUB01	Left	Moderate	30%
SUB02	Bilateral (right > left)	Mild	100% (right); and 30% (left)
SUB03	Right	Catastrophic	0% (residual excitation)
SUB04	Left	Slight	25%
SUB05	Right	Mild	25%
SUB0	Right	Mild	30%
SUB07	Bilateral (left > right)	Mild	100% in both ears
SUB08	Left	Mild	50%
SUB09	Bilateral (right > left)	Slight	25% (right = lrft)
SUB10	Left	Mild	0%

Abbreviations: SUB, subject; THI, Tinnitus Handicap Inventory.

Subjective Measures

The subjective outcome measures of all the participants are summarized in ►**Table 2**. Overall, 90% observed a change in the tinnitus loudness after undergoing psychoeducational counseling and RIT. In total, 8 out the the 10 participants had residual inhibition, 1 (SUB 03) had residual excitation, and 1 (SUB 10) had no change.

Objective Measures: (CAEP Results)

The means and standard deviations of the latency of N1 and P2 and the amplitude before anf after the tinnitus treatment are shown in ►**Table 3**. The repeated mesures ANOVA showed no significant main effect of the electrode (F [1, 9] = 0.748; $p = 0.410$; $\eta^2 = 0.077$) and the treatment (F [1, 9] = 0.002; $p = 0.964$; $\eta^2 = 0.000$) on the latency of N1,. neither was their interaction significant (F [1, 9] = 0.987; $p = 0.346$; $\eta^2 = 0.099$). There was a significant main effect of the treatment on the amplitude of N1 (F [1, 9] =19.088; $p = 0.002$; $\eta^2 = 0.680$), which was was reduced after the treatment (►**Table 3**). The amplitude of N1 also differed significantly regarding electrode sites (F [1, 9] = 6.159; $p = 0.035$; $\eta^2 = 0.406$). There was no significant interaction effect (F [1, 9] = 1.375; $p = 0.271$; $\eta^2 = 0.133$). The repeated measures ANOVA showed no significant main effect of the electrode sites (F [1, 9] = 1.699; $p = 0.225$; $\eta^2 = 0.159$) or of the treatment (F [1, 9] = 0.140; $p = 0.717$; $\eta^2 = 0.015$) on the latency of P2. The interaction between the electrode sites and the treatment was not significant (F [1, 9] = 0.234; $p = 0.640$; $\eta^2 = 0.025$). The statistical results for the amplitude of P2 were similar to those of the latency: there was no significant main effect of the electrode sites (F [1, 9] = 0.000; $p = 0.989$; $\eta^2 = 0.000$) and of the treatment (F [1, 9] = 1.425; $p = 0.263$; $\eta^2 = 0.137$). Moreover, the interaction effect regarding the treatment and electrode was not significant (F [1, 9] = 1.161; $p = 0.698$; $\eta^2 = 0.018$).

P3 Component

The mean and standard deviation of the latency and amplitude of P3 before and after the tinnitus treatment are shown in ►**Table 3**. There was no significant main effect of the treatment (F [1, 9] = 0.005; $p = 0.943$; $\eta^2 = 0.001$) on P3 latency. The results showed that there was a main effect of the electrode sites (F [2, 18] = 5.949; $p = 0.010$; $\eta^2 = 0.398$) on P3 latency, but no interaction between the electrodes and the treatment (F [2, 18] = 0.227; $p = 0.799$; $\eta^2 = 0.025$). The analysis of the amplitude of P3 showed a significant main effect of the treatment (F [1, 9] =15.40; $p = 0.003$; $\eta^2 = 0.631$): after the treatment, there was a reduction in the amplitude of P3 compared to before the treatment. There was no significant main effect of the electrodes (F [2, 18] =1.332; $p = 0.289$; $\eta^2 = 0.129$), but the interaction between the electrodes and the treatment was significant (F [2, 18] = 7.816; $p = 0.004$; $\eta^2 = 0.465$).

Correlation between Subjective and Objective Measures

The Pearson correlation was performed to assess the relationship between the perceived difference in tinnitus loudness and P3 amplitude (at Pz). There was no correlation between the two variables ($r = -0.575$; $n = 10$; $p = 0.082$), which suggests no correlation between the reduction in P3 amplitude (at Pz) and the perceived loudness. The scatter plot summarizing the results is provided in ►**Fig. 2**.

Comparison with the Historical Control Group

The historical control group[25] was matched in terms of age (t [18] = 0.194; $p = 0.848$), gender and hearing (right PTA: t [18] = 2.011; $p = 0.060$; left PTA: t [18] = 1.882; $p = 0.076$) to the case group. The mean latencies and amplitudes of the historical control group are shown in ►**Table 3**. The analysis of the posttreatment CAEPs and the historical control group using repeated measures ANOVA indicated that the latencies (N1: F [1, 18] = 0.006; $p = 0.939$; $\eta^2 = 0.000$; P2: F [1, 18] = 1.227; $p = 0.283$; $\eta^2 = 0.064$; and P3 F [1, 18] = 0.726; $p = 0.405$; $\eta^2 = 0.039$) and amplitudes (N1: F [1, 18] = 3.288; $p = 0.086$; $\eta^2 = 0.154$; P2: F [1, 18] = 0.808; $p = 0.380$; $\eta^2 = 0.043$; and P3 F [1, 18] = 0.192; $p = 0.667$; $\eta^2 = 0.011$) of the ERPs were comparable between the two groups (►**Figs. 3** and **4**).

Table 3 Means and standard deviations of the latency and amplitude of the N1, P2, and P3 components

Component	Group	mean latency (± standard deviation) (in ms)			*p*-value	mean amplitude (± standard deviation) (in μV)			*p*-value
		Fz	Cz	Pz		Fz	Cz	Pz	
N1	**Tinnitus (Pretreatment)**	97.20 (± 15.55)	99.10 (± 13.53)	–	0.964	-2.65 (± 1.89)	-1.44 (± 1.13)	–	0.002*
	Tinnitus (Posttreatment)	102.3 (± 10.42)	100.50 (± 13.18)	–		-1.84 (± 1.06)	-0.97 (± 0.93)	–	
	Control	101.90 (± 12.77)	101.30 (± 11.36)	–	Postreatment versus control: 0.939	- 0.90 (± 1.28)	-0.73 (± 0.59)	–	Post ± reatment versus control: 0.086
P2	**Tinnitus (Pretreatment)**	169.60 (± 19.06)	174.20 (± 21.85)	–	0.717	0.61 (± 1.66)	1.04 (± 0.62)	–	0.263
	Tinnitus (Posttreatment)	179.80 (± 24.69)	179.10 (± 20.81)	–		0.54 (± 1.51)	1.09 (± 0.67)	–	
	Control	171.00 (± 19.11)	172.70 (± 17.32)	–	Posttreatment versus control: 0.283	1.41 (± 1.20)	1.05 (± 1.13)	–	Posttreatment versus control: 0.380
P3	**Tinnitus (Pretreatment)**	350.90 (± 28.25)	371.10 (± 39.99)	386.40 (± 25.22)	0.943	1.657 (± 2.98)	3.36 (± 2.03)	4.22 (± 2.04)	0.003*
	Tinnitus (Posttreatment)	354.60 (± 25.63)	363.80 (± 35.31)	387.90 (± 22.01)		1.18 (± 2.78)	1.55 (± 1.85)	1.857 (± 2.25)	
	Control	341.80 (± 23.65)	361.40 (± 21.54)	384.20 (± 22.79)	Posttreatment versus control: 0.405	0.52 (± 2.80)	2.38 (± 1.93)	2.54 (± 1.65)	Posttreatment versus control: 0.667

Note: *Statistically significant.

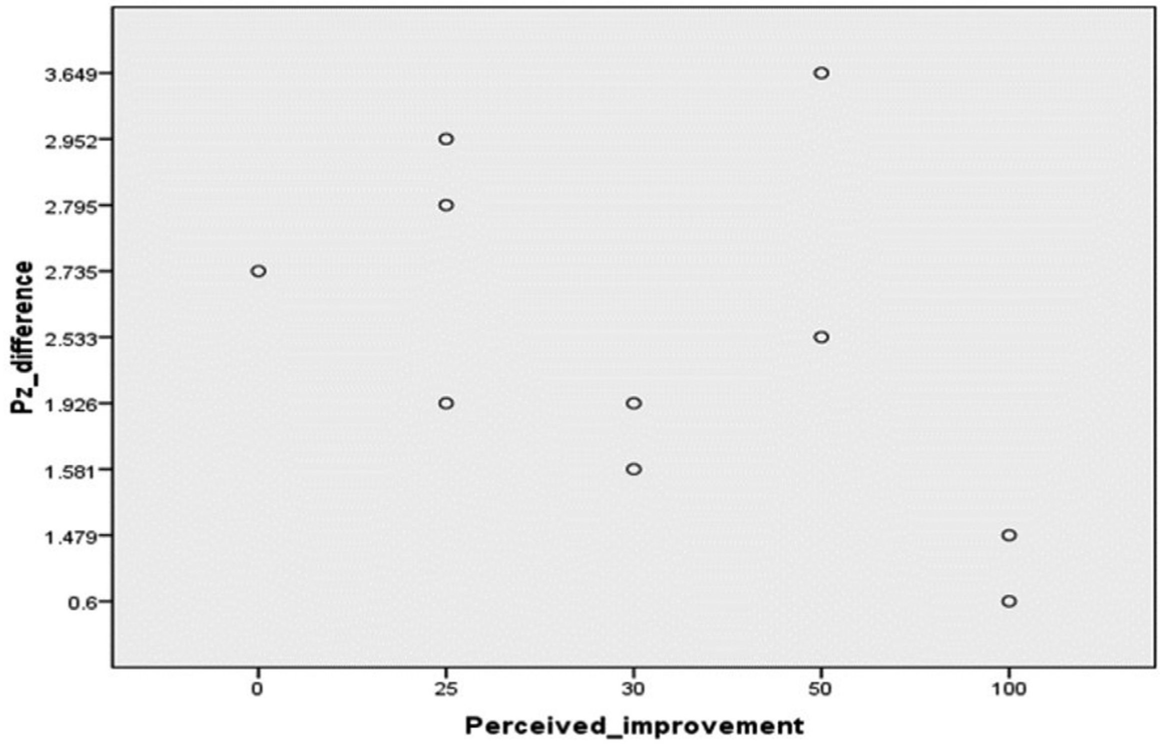

Fig. 2 Scatter plot (correlation between the difference in P3 amplitude at Pz and the perceived reduction in loudness).

Discussion

Residual inhibition is a commonly-used procedure, but there are very few studies on the mechanism of RI to relieve tinnitus. The present study aimed at exploring whether CAEPs reflected the effect of RIT and educational counselling using the N1 and P3 auditory components and their correlation with behavioral measures. Overall, the results of the present study reflected both subjective and objective changes in individuals with tinnitus after a single session of RIT and psychoeducational counseling. The results are discussed in the following sections.

Subjective Measures

The literature[33,34] supports a reduction in the severity of the tinnitus after RI to different stimuli. The present study is in line with these findings: 80% of the participants reported a reduction in tinnitus loudness after a session of modified RIT and psychoeducational counselling. However, SUB03 and SUB10 reported no change in loudness. It is noteworthy that SUB03 reported a catastrophic handicap due to tinnitus in the THI. The duration of RI for the other eight participants was of at least one hour (until the participants were in the hospital for the posttreatment evaluation). The participants were informed that the effect may be temporary and can ricochet. They were advised not to panic in case the tinnitus reoccured, and to reach out to us in such situations. All participants were counselled on environmental modifications based on their RIT results and future tinnitus care. None of the participants returned complaining of tinnitus.

Objective Measures

N1 Component

The results of the present study show a significant reduction in the amplitude of N1 after the treatment. The pre- and posttreatment N1 waveforms are shown in ►**Fig. 3**.

It is known that individuals with tinnitus have a larger N1 amplitude.[25,35] Enhanced neural synchrony resulting in higher baseline activity or an impaired adaptation of cortical neurons following noise exposure/cochlear damage are thought to be the underlying mechanisms. Residual inhibition directly influences the neural firing of cortical neurons by reducing the neuronal hyperexcitability in animals suffering from tinnitus.[16] The amplitude of N1 indexes the strength of the neural firing. Hence, it can be deduced that the reduction in N1 amplitude can be related to neuronal silencing. With the assessments conducted in the present study, we may not be able to pinpoint the exact mechanism triggering this change. Nevertheless, the change in the sensory component indicates an unambiguous plastic change at the cortical level after a single session of modified RIT and psychoeducational counseling. N1 latency indexes the speed of stimulus detection or the conduction delay. Concerning N1 latency, there was no observable change after RIT and psychoeducational counselling, which implies that the treatment does not alter neuronal efficiency. The N1 reduction (at Fz) for the individual subjects is shown in ►**Fig. 5**. Eight out of the ten participants showed a clear reduction in their N1 amplitude posttreatment. Overall, the results regarding N1 suggested a measurable difference before and after a single session of tinnitus treatment. It is

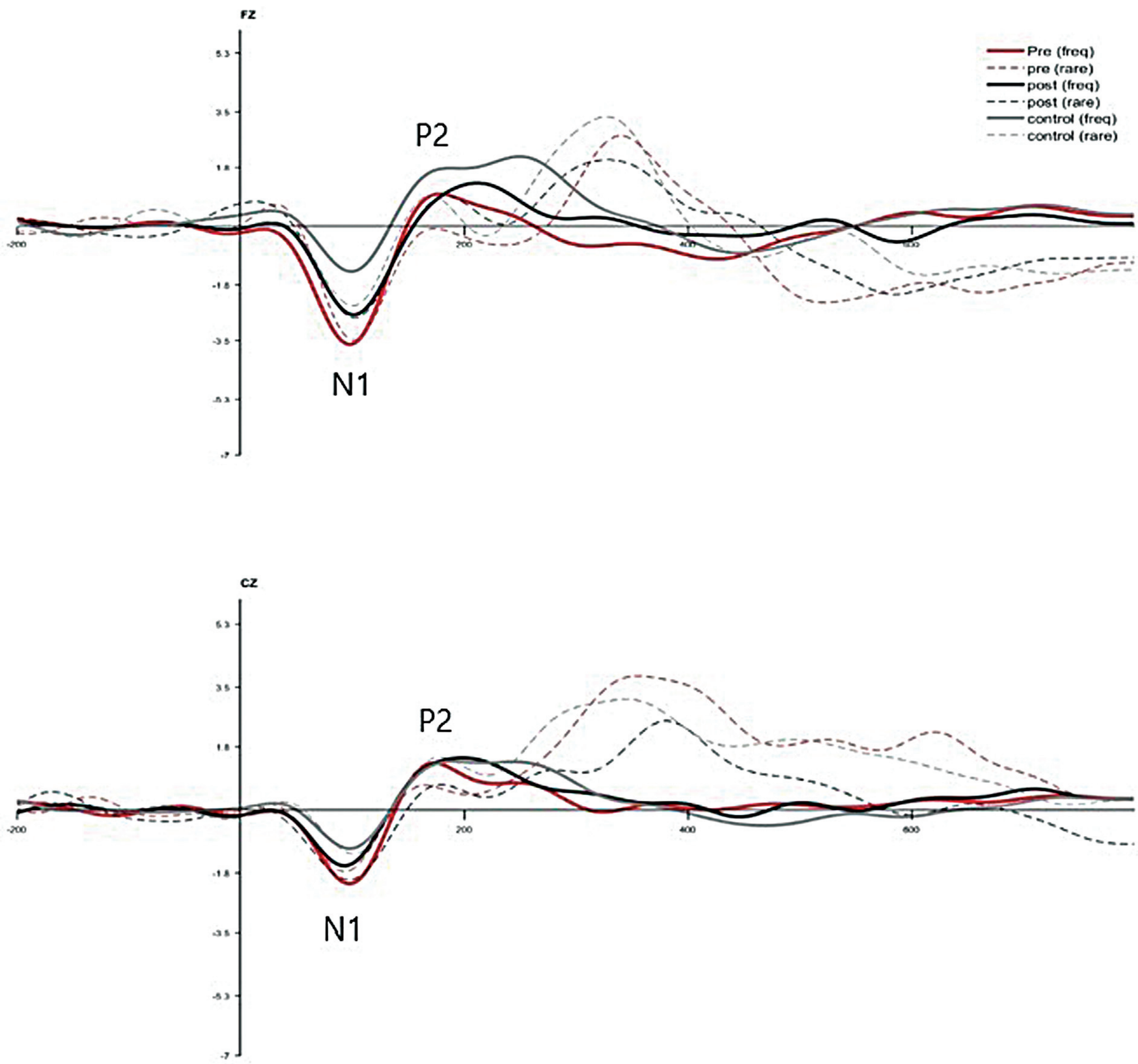

Fig. 3 N1 waveform pre- and posttreatment with results of the control group as well.

plausible that this change in N1 is a result of neuronal silencing contributing to tinnitus reduction. However, considering that not all the participants showed a reduction in N1 amplitude and the sample size of the study, these findings must be interpreted with caution.

P3 Component

The current study showed a reduction in P3 amplitude after RIT and psychoeducational counselling without any latency change. The pre- and posttreatment P3 waveforms are shown in ►**Fig. 4**.

Individuals with tinnitus typically showed a mixed presentation, with associated hearing loss and psychological problems. These factors affect P3 in their ways, making it difficult to interpret. P3 amplitude is an index of central resource allocation and working memory processing. The literature supports that individuals with tinnitus usually have a decreased[16,20,23] or unaffected P3 amplitude.[36] Similar findings are observed in individuals with pure sensory neural hearing impairment.[37,38] Such a decrease in P3 amplitude was related to insufficient resource allocation and/or working memory. However, individuals with psychological ailments (including phobia and anxiety) have been shown to have an increased P3 amplitude.[39–44] Increased attentional resources getting allocated for a target stimulus can increase the activity in the ventral attentional network (similar to anxiety disorders) which in turn can result in an enhanced P3.[43] It is well known that individuals with tinnitus have increased anxiety and fear due to the involvement of the limbic and autonomic nervous systems.

Objective differences in the neuronal oscillatory bands have been found after an RI session.[45–47] In the present study, there was a reduction in P3 amplitude posttreatment. It is only plausible to expect that the fear and anxiety associated with tinnitus would have reduced post treatment which is projected via the P3 reduction. Besides, RIT is known to affect neuronal excitability[16] by causing neuronal silencing, triggering a temporary relief of the tinnitus. This temporary relief could have made the resource allocation simpler. The combined efforts of these mechanisms might have resulted

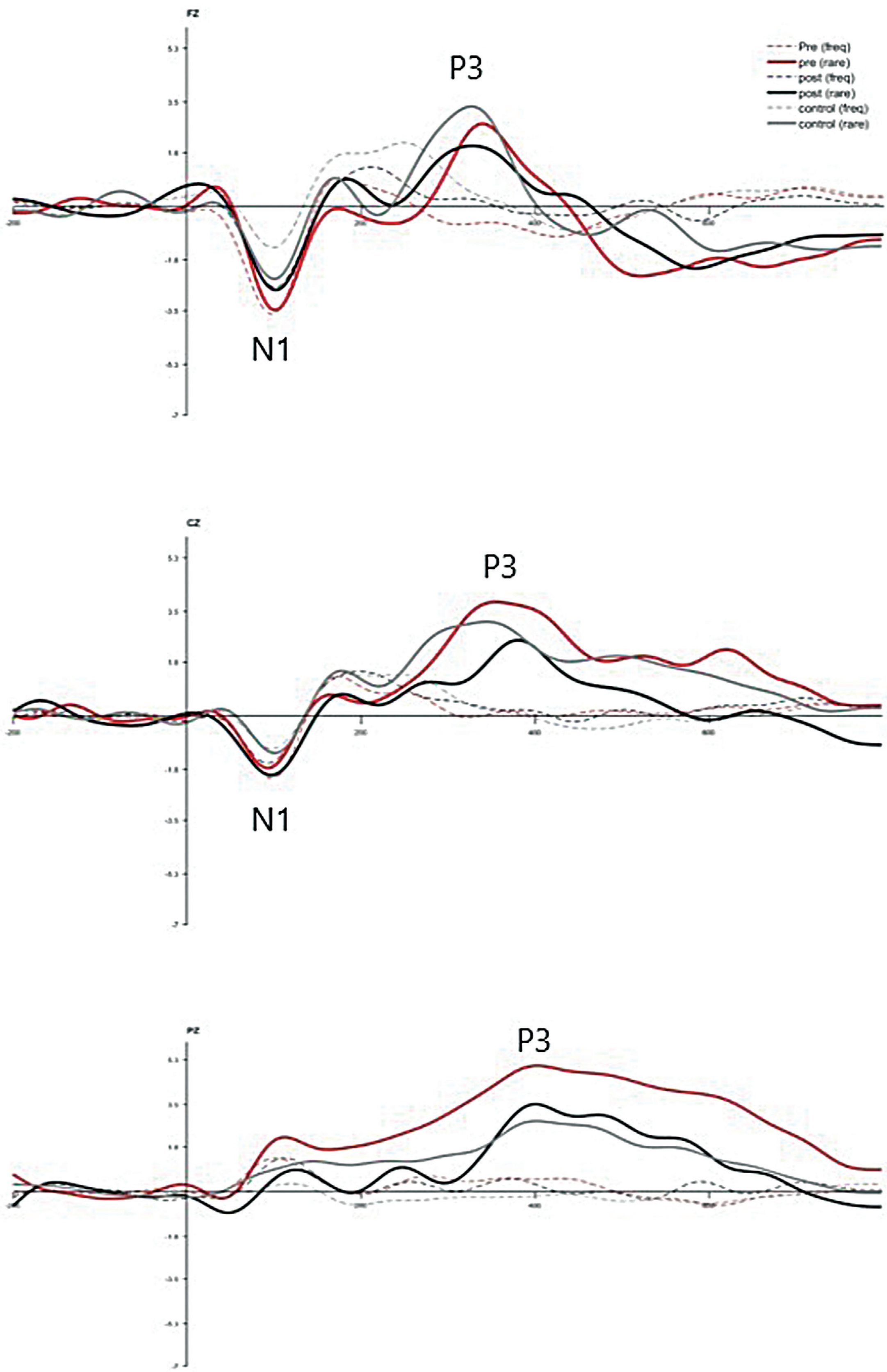

Fig. 4 P3 waveform pre- and posttreatment with results of the control group as well.

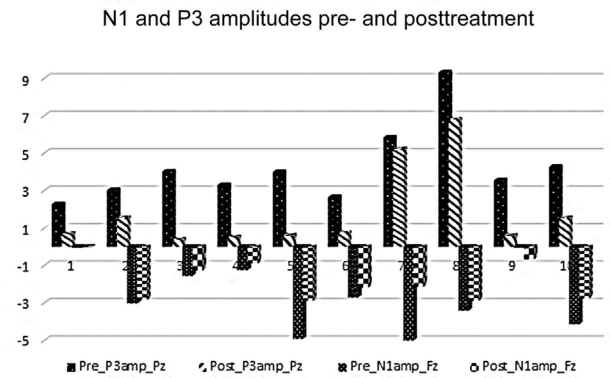

Fig. 5 Pre- and posttreatment N1 and P3 amplitudes for the individual participants

in a moderated P3 amplitude, which was increased before the treatment.[25] Interestingly, the posttreatment P3 amplitudes were similar to those of the historical control group. However, since the participants performed the same task twice (before and after the treatment), the effect of practice cannot be neglected. In addition, since psychological factors related to tinnitus, such as anxiety and fear, were not measured using standardized scales, it is difficult to attribute the decrease in P3 to the impact of the psychological factors.

►**Fig. 5** displays the reduction in P3 amplitude for each individual after the treatment, and it shows that the reduction was unidirectional for all participants rather than just a group effect. A reduction of 2 µV to 3 µV in P3 was observed in all participants at the Cz and Pz electrode sites. Unlike the sensory component N1, the cognitive component P3 was found to be reduced in all the participants after the treatment, indicating that RIT and psychoeducational counseling not only influence the neuronal excitability but also has an influence on the central cognitive components of processing.

In the present study, no modification was observed on P3 latency posttreatment. The P3 latency indexes information processing and the speed of classification of a stimulus, and it is reported[21,22] to be longer in individuals with tinnitus; however, there are contradictory findings[36,48] as well. A possible explanation for the lack of alteration in latency in the current study could be that RIT does not target the working memory, subsequently causing the efficiency of the stimulus classification to remain unaltered.

Correlation between Subjective and Objective Measures

In the present study, there was a reduction in both the perceptual (VAS) and functional (CAEPs) measures posttreatment. A decrease in tinnitus loudness could reduce the cognitive load, as discussed earlier. However, no significant correlation was observed between the reduction in P3 amplitude and the reduction in the perceived tinnitus loudness, though both of these measures changed in the same direction. This could be attributed to the small sample size or the mechanism underlying these measures, or both. P3 reflects the cognitive resources allocated to process a particular stimulus and working memory, which is one of the stages that is involved in perception. Loudness is the psychological attribution of the intensity of a sound, which is an end product of multiple decision making process.

Limitations of the Study

First, the sample size of the present study makes generalization tricky. Nevertheless, it still comprises preliminary evidence in favor of psychoeducational counselling and RIT. Second, a follow-up evaluation of the participants would have provided more information on the duration of this effect of the treatment. This was not possible due to various reasons (including travel, distance, and financial issues involving the participants), making it a drawback of the study. Finally, the study included two individuals who did not fall within the persistent tinnitus category. Though the current study is a pre-post design the different categories of tinnitus could still be a confounding factor which needs to be studied further.

Conclusion

There was a significant reduction in the amplitude of both the sensory (N1) and cognitive (P3) ERP components, with no alteration in their latencies after psychoeducational counselling and RIT, possibly reflecting both perceptual and functional changes (evidenced as reduced N1 and P3). This is preliminary evidence that ERPs can be used to quantify the physiological changes that occur after tinnitus intervention. However, further randomized clinical trials with a large samples are warranted to establish this.

Highlights

- A single session of modified RIT and psychoeducational counselling can bring about perceptual and functional changes at the cortical level.
- Event-related potentials (ERPs) may serve as a tool to quantify functional changes after tinnitus treatment.

Funding
The present research did not receive any specific grants from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interests
The authors have no conflict of interests to declare.

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